AGE-STRUCTURED CORE GROUP MODEL AND ITS IMPACT ON STD DYNAMICS

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Age-structured Core Group Model and Its Impact on STD Dynamics

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Abstract The recruitment of new suceptibles into a core group of sexually-active individuals may depend on the current levels of infection within a population. We extend the formalism of Hadeler and Castillo-Chavez (1995), that includes prevalence dependent recruitment rates, to include age structure within core and noncore populations. Some mathematical results are stated but only a couple of proofs are included since our objetives are to highlight the modeling process and the dynamic possibilities. This paper concludes with an example where endemic distributions can be supported when the basic reproductive number R_0 is less than one. Systems that are capable of supporting multiple attractors are more likely to support disease re-emergence. This model is likely to support stable multiple attractors when $R_0 < 1$.

0. Introduction

Core groups are generally small subpopulations capable of supporting strong transmission rates and high disease prevalence. Effective disease management strategies not only cannot ignore core groups but in fact must focus on them (Hethcote and Yorke 1984). The role of behavior on the dynamics of sexually transmitted disease (STDs) gained additional importance with the emergence of HIV, in western societies, nearly two decades ago (Castillo-Chavez 1989; Hethcote and Van Ark, 1992). Important changes in behavior were observed and documented in various homosexually-active populations including those living in San Francisco, New York and Boston. These observed changes included a reduction in average sexual activity, a decline in risky behavior, and a decrease in the rate of unprotected sexual contacts (see Baldwin and Baldwin 1988, Curran et al. 1988, Fineberg 1988, Evans et al. 1989, Martin 1987, Saltzman et al. 1987, Shechter et al. 1988, van Griensven et al. 1989a,b, Wilkenstein 1988, and Wiktor et al. 1990). Recent data (see below) seem to support the view that at best these changes were not as prevalent as one would have liked to and at worst they may have been kept only for a short period of time.

The combined effects of short- and long-term reductions of risk behaviors on the transmission dynamics of sexually-transmitted diseases (STDs) has been explored in limited ways via the use of simple models (Blythe et al. 1993a, 1993b, 1997; Brauer et al. 1998; Hadeler and Castillo-Chavez, 1995; Hsieh and Cooke 2000; Velasco et al. 1996; and Velasco and Hsieh, 1994). The theoretical results and predictions obtained via mathematical models have helped define a nontrivial landscape (based on the qualitative dynamics of these models) in which appropriate evaluations of the effectiveness of disease management strategies may be tested. An effective program of evaluation must be capable of tracing the origin of behavioral changes. If changes are long term and the result of a systematic implementation of educational efforts then the program would be rated as highly effective. However, whenever such changes are temporary then the effectiveness of such program must necessarily be questioned. Educational programs that "work" only when disease prevalence is high are not effective since its "effectiveness" may decrease when individuals' risk and fear of infection goes down. Hadeler and Castillo-Chavez (1995) showed that educational programs "may not only fail to have a positive impact in core populations with low disease prevalence but may

even increase their effective group size by implying a reduction in risk within the core."

Erin McClam from the *Associated Press* in her article "Syphilis outbreak alarms officials" (*Ithaca Journal*, February 23, 2001, page $10A$) highlights the criticality of long-term behavioral changes. She cites several authorities: "HIV 'is no longer perceived to be the threat that it once was' said Dr. Ronald Valdeserri"... In fact, "Syphilis outbreaks in major cities 'show that the disease can make a comeback,' said Ken August'..." Ms. McClam includes some local statistics that show that out of the last 66 (from a total of 130) syphilis cases reported among gay men during the first semester of 2000 in four California Counties, "...33 reported they had anonymous sex, and 17 said they had met sex partners in bathhouses. Only one in five reported using a condom during his most recent sexual encounter, and two in five reported using illegal drugs." The connection to temporal changes in behavior and current policy is clear from Valdiserri's statement " ...'When we see reports of increasing risk behaviors, that's when we take action. We don't wait till we see the spike nationally' ..." Finally, it was noted in Ms. McClam's article that 34 out of 66 men who had syphilis were also HIV-positive while 9 of them had no idea about their HIV status. The memory of the fatality rates associated with HIV on homosexually-active men in San Francisco and similar cities seemed to have lost its impact on current generations of homosexually-active gay men. One of the possible consequences associated with such memory loss is the remergence of HIV (as predicted by Hadeler and Castillo-Chavez, 1995).

This paper is organized as follows: Section 1 introduces an age-structure model with prevalence dependent recruitment rates; Section 2 looks at the local stability of the infection-free distribution; Section 3 states the conditions for the existence of nonuniform endemic age distributions and gives an example where endemic age distributions are possible even though $R_0 < 1$. The occurrence of a backward (subcritical) bifurcation, several infected stationary states, and hysteresis phenomena (including abrupt changes in disease prevalence levels) may be possible in this model. Section 4 states our conclusions.

1. The model

Here we focus on disease dynamics within a core group population that recruits individuals from a non-core population. It is assumed that the rate of recruitment r

from the non-core into the core depends on the level of disease prevalence within the core group. Specifically, it is assumed that recruitment slows down when prevalence is high and increases when it is low. The epidemic process is built on a simple demographic setting. It is assumed that the non-core group is completely inactive (no sexual activity), that is, it just serves as the source of new members for the core. It is further assumed that the average contact rate in the core group is age-dependent $B(a)$, that infected individuals are symptomatic, and that infected individuals may return (at a constant per capita rate γ) to the susceptible class after treatment. Individuals are assumed to mix at random (proportionate mixing; Hethcote and Van Ark, 1987; Busenberg and Castillo-Chavez, 1991 or Castillo-Chavez et al. 1994) and the per capita mortality rate (μ) is assumed to be constant (independent of age). The model is formulated as a general homogeneous system (Hadeler et al. 1988, Hadeler 1992, Busenberg and Hadeler 1990) but only the case where the total population is either constant or asymptotically constant is discussed.

We let $q(a,t)$ denote the non-core and $c(a,t)$ the core group age-dependent densities. We sub-divide the core group into two epidemiological classes: susceptibles $s(a,t)$ and infectives $u(a,t)$. Let $r = r(\frac{U(t)}{C(t)})$ denote the per capita recuitment rate of sucetibles from the non-core (outside group with density $p(a,t)$) where $C(t) = \int_0^\infty c(t,a)da = \int_0^\infty [s(t,a) + u(t,a)]dt$ and $U(t) = \int_0^\infty u(t,a)da$. That is, $C(t)$ and $U(t)$ denote the total core group population and the population of infectives (within the core group) at time t, respectively. The disease dynamics in this setting can be described by the following set of equations:

$$
\begin{aligned}
(\frac{\partial}{\partial a} + \frac{\partial}{\partial a})q(t,a) &= -\mu q(t,a) - r(\frac{U(t)}{C(t)})q(t,a), \\
(\frac{\partial}{\partial t} + \frac{\partial}{\partial a})s(t,a) &= -\mu s(t,a) + \gamma u(t,a) + r(\frac{U(t)}{C(t)})q(t,a) - \beta B(a)s(t,a)\Lambda(t,a), \\
(\frac{\partial}{\partial t} + \frac{\partial}{\partial a})u(t,a) &= -(\mu+\gamma)u(t,a) + \beta B(a)s(t,a)\Lambda(t,a),
\end{aligned}
\tag{1.1}
$$

with boundary conditions

$$
q(t,0) = \mu K, \qquad s(t,0) = u(t,0) = 0, \tag{1.2}
$$

and initial conditions

$$q(0,a) = q_0, \qquad s(0,a) = s_0, \qquad u(0,a) = u_0.$$

$B \in L^\infty(\mathbb{R}^+)$ is nonnegative, $\lim_{a\to\infty} B(a) = 0$,

$$\Lambda(t,a) = \frac{\int_0^\infty B(s)u(t,s)ds}{\int_0^\infty B(s)c(t,s)ds},$$

and q_0, s_0 and $u_0 \in L^1(\mathbb{R}^+)$ are all nonnegative. Furthermore, it is assumed that $r(v)$ is decreasing and positive for $v \in [0,1]$. Since $p(t,a) = q(t,a) + s(t,a) + u(t,a)$ is the density of the total population then from the addition of the three equations in (1.1) we see that $p(t,a)$ obeys the classical age-structured model:

$$(\frac{\partial}{\partial t} + \frac{\partial}{\partial a})p(t,a) = -\mu p(t,a), \qquad p(t,0) = \mu K. \tag{1.3}$$

The integration of Equation (1.3) along characteristic lines gives

$$\lim_{t\to\infty} p(t,a) = \mu K e^{-\mu a}, \quad a \geq 0,$$

that is, $p(t,a)$ converges to its stable age-dsitribution.

In order to simplify the analysis it will be assumed throughout this paper that the total population $p(t,a)$ has reached its stable distribution, that is, that

$$p(t,a) = p(a) = \mu K e^{-\mu a}, \quad a \geq 0.$$

Hence, it is implicitly assumed that the disease is nonfatal.
The substitution of q by $Ke^{-\mu a} - c$, leads, using the second and third equations in (1.1), to the qualitatively equivalent simplified model

$$\begin{aligned}
(\frac{\partial}{\partial t} + \frac{\partial}{\partial a})c(t,a) &= -\mu c(t,a) + r(\frac{U(t)}{C(t)})(\mu K e^{-\mu a} - c), \\
(\frac{\partial}{\partial t} + \frac{\partial}{\partial a})u(t,a) &= -\sigma u(t,a) + \beta B(a)(c(t,a) - u(t,a))\Lambda(t,a),
\end{aligned} \tag{1.4}$$

where $\sigma = \mu + \gamma$. The rest of this paper focuses on the study of the role of the basic reproductive number, R_0, on the local stability of the disease free distribution

and on the possible existence of multiple (endemic) equilibria for System (1.4) (see Castillo-Chavez et al. 2001; this volume).

2. Infection-free distribution and the thresold condition

System(1.4) always admits the disease-free distribution $(c^0, 0)$ as a solution, where c^0 is the positive solution of the differential equation

$$\dot{c}(a) = -\mu c(a) + r(0)(\mu K e^{-\mu a} - c(a)), \qquad 0 \leq a \leq \infty, \tag{2.1}$$

with initial condition $c(0) = 0$. Thus c^0 is uniquely given by the explicit expression

$$c^0(a) = \int_0^a e^{-(\mu + r_0)(a-\theta)} \mu K e^{-\mu\theta} d\theta = \mu K e^{-\mu a}(1 - e^{-r_0 a}),$$

where $r_0 = r(0)$.

We now turn to study the stability of the disease free distribution. A straightforward computation shows that the linearized system at the disease free distribution is given by the system:

$$\begin{aligned} (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})x(t,a) &= -(\mu + r_0)x(t,a) + \nu(a)y(t,a), \\ (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})y(t,a) &= -\sigma y(t,a) + \alpha(a)\int_0^\infty B(s)y(t,s)ds, \\ x(t,0) &= x(t,0) = 0, \end{aligned} \tag{2.2}$$

where

$$\nu(a) = \frac{r'(0)\mu K e^{-\mu a}}{C}, \qquad \alpha(a) = \frac{\beta B(a) c^0(a)}{\int_0^\infty B(\tau)c^0(\tau)d\tau}.$$

It is simple to show that the reproductive number (see Castillo-Chavez et al. 2001; this volume) is given by

$$R_0 = \int_0^\infty \int_0 B(a) e^{-\sigma(a-\tau)} \alpha(\tau) d\tau da.$$

The basic reproductive number R_0 (see Diekmann et al. 1990; Diekemann and Heesterbeek, 2000; and Castillo-Chavez et al. 2001) is used to settle the question of local asymptotic stability.

Theorem 2.1 *If $R_0 < 1$ and if B is uniformly Lipshitz continuous on $\mathbb{R}^+$ then the disease free equilibrium is locally asymptotically stable.*

Proof: It is enough to show that the linear system (2.2) is asymptotically stable. First consider the second equation of (2.2). Let $S(t), t \geq 0$ be the semigroup corresponding to this equation and let $T(t), t \geq 0$ be a semigroup defined by the system

$$(\frac{\partial}{\partial t} + \frac{\partial}{\partial a})y(t,a) = -\sigma y(t,a),$$

$$y(t,0) = 0, \quad y(0,\cdot) = y_0 \in L^1(\mathbb{R}^+).$$

Since B is uniformly Lipshitz continuous and $\lim_{a\to\infty} B(a) = 0$ then by the definition of $\alpha(a)$ it can be verified that the operator $L : L^1(\mathbb{R}^+) \to L^1(\mathbb{R}^+)$:

$$[L\phi](a) = \alpha(a) \int_0^\infty B(s)\phi(s)ds$$

is compact. Therefore, it follows from the variation-of-constants formula that $S(t)$ is a compact perturbation of $T(t)$. Hence (see G.F. Webb, Proposition 4.1, p.178-179; 1985) $\gamma_{E\sigma}(T(t)) = \gamma_{E\sigma}(S(t))$, where $\gamma_{E\sigma}(L)$ denotes the radius of the essential spectrum of an operator L. Moreover, it is known (see, Webb, Example 4.10, p.175-176; 1985) that

$$\gamma_{E\sigma}(T(t)) = e^{-\sigma t}.$$

Now we consider the eigenvalues of the infinitesimal generator A of the semigroup $S(t)$ given by

$$[A\phi](a) = -\dot{\phi}(a) - \sigma\phi(a) + \alpha(a) \int_0^\infty B(s)\phi(s)ds,$$

with

$$\mathcal{D}(A) = \{\phi \in L^1(\mathbb{R}^+) : \dot{\phi} \in L^1(\mathbb{R}^+), \phi(0) = 0\}.$$

If λ is an eigenvalue of A and ϕ its corresponding eigenvector then

$$\dot{\phi}(a) = -(\lambda + \sigma)\phi(a) + \alpha(a) \int_0^\infty B(s)\phi(s)ds.$$

Hence

$$\phi(a) = \int_0^a e^{-(\lambda+\sigma)(a-\theta)}\alpha(\theta) \int_0^\infty B(s)\phi(s)ds,$$

that is,

$$|\phi(0)| \leq \int_0^a e^{-(\Re\lambda+\sigma)(a-\theta)}\alpha(\theta)\int_0^\infty B(s)|\phi(s)|ds.$$

Multiplying both sides of the above inequality by $B(a)$ and integrating it over the interval $[0,\infty)$ gives

$$1 \leq \int_0^\infty B(a)\int_0^a e^{-(\lambda+\sigma)(a-\theta)}\alpha(\theta)d\theta da. \tag{2.3}$$

Since

$$R_0 = \int_0^\infty B(a)\int_0^a e^{-\sigma(a-\theta)}\alpha(\theta)d\theta da < 1,$$

then Inequality (2.3) implies that there is a number $w_0 > 0$ such that $\Re\lambda \leq -w_0$. It therefore follows that

$$\sup\{\Re\lambda : \lambda \in \sigma_p(A)\} \leq -w_0.$$

Hence, letting $\xi = \min\{\sigma, w_0\}$ implies the existence of an $M > 0$ such that $\|S(t)\| \leq Me^{-\xi t}$, for all $t \geq 0$. Therefore,

$$y(t,\cdot) \leq M\|y_0\|_{L^1}e^{-\xi t}, \quad t \geq 0. \tag{2.4}$$

The first equation of (1.5) and the variation-of-constants formula implies that there exists a semigroup $T_1(t)$ such that $\|T_1(t)\| \leq e^{-(\mu+r_0)t}$, $t \geq 0$ and such that

$$x(t,\cdot) = T_1(t)x_0 + \int_0^t T_1(t-s)\nu(\cdot)y(s,\cdot)ds. \tag{2.5}$$

From (2.4) and (2.5) we conclude that there is a positive number M_1 such that

$$\|x(t,\cdot)\| \leq \|x_0\|e^{-(\mu+r_0)t} + M_1\|y_0\|(e^{-\xi t} + e^{-(\mu+r_0)t}).$$

Hence System (1.5) is asymptotically stable.

3. Existence of Endemic Equilibria

This section outlines the steps required in the proof of the existence of an endemic equilibrium when $R_0 > 1$. That is, we study the existence of positive solutions of the

system of differential-integral equations

$$\begin{aligned} \dot{c}(a) &= -\mu c(a) + r(\frac{U}{C})(\mu K e^{-\mu a} - c(a)) \\ \dot{u}(a) &= -\sigma u(a) + \frac{\beta B(a)(c(a) - u(a)) \int_0^\infty B(s)u(s)ds}{\int_0^\infty B(s)c(s)ds} \\ c(0) &= u(0) = 0, \end{aligned} \tag{3.1}$$

where $U = \int_0^\infty u(a)da$, $\int_0^\infty C = \int_0^\infty c(a)da$.
Since $0 \leq c(a) \leq p(a) = \mu K e^{-\mu a} \to 0$ as $a \to \infty$ and $c(0) = 0$ then the integration of both sides of (3.1) gives

$$\mu C = r(\frac{U}{C})(K - C),$$

or equivalently

$$r(\frac{U}{C}) = \frac{\mu C}{K - C}. \tag{3.2}$$

In our search for a positive solution of (3.1) C is treated as a free parameter with $C \in (0, K)$ and the function

$$f(C) = \frac{\mu C}{K - C}, \quad 0 < C < K$$

is considered.
Whenever (c, u) is a solution of (3.1) then use of the first equation of (3.1) gives

$$c(a) = h(C, a) := \mu K e^{\mu a}(1 - e^{-f(C)a}).$$

Hence, a positive solution of (3.1) must be a solution of the following differential-integral equation

$$\begin{aligned} \dot{u}(a) &= -\sigma u(a) + \frac{\beta B(a)}{\int_0^\infty B(s)h(C, s)ds}[h(C, a) - u(a)] \int_0^\infty B(s)u(s)ds, \\ u(0) &= 0, \end{aligned} \tag{3.3}$$

subject to the constraint

$$r(\frac{\int_0^\infty u(a)da}{\int_0^\infty h(C, a)da}) = f(C). \tag{3.4}$$

The proof of the existence of positive solutions is technical and requires the proof of a series of lemmas. Here we list these lemmas without proof (see Huang and Castillo-Chavez 2001).

Lemma 3.1 *Let* $G(C,a) = \dfrac{\beta B(a)}{\int_0^\infty B(s)h(C,s)ds}$. *If we assume that* $B(a) > 0$ *for all* $a \geq 0$ *then the differential-integral equation (3.3) has a positive solution* $u_C \leq h(C,\cdot)$ *if and only if*

$$\int_0^\infty B(a) \int_0^a e^{-(\mu+\gamma)(a-s)} G(C,s)ds > 1.$$

Furthermore, the solution u_C *depends continuously on* C.

Lemma 3.2 *If* $u_C > 0$ *is a solution of (3.2) then there is a* $0 < \rho < 1$ *such that for each* $0 < C \leq C_0$

$$\frac{U_C}{C} = \frac{\int_0^\infty u_C(a)da}{C} \leq \rho.$$

Theorem 3.3 *If* $\int_0^\infty B(a) \int_0^a e^{-(\mu+\gamma)(a-s)} G(C_0,s)ds > 1$ *and* $B(a)$ *is strictly positive then (3.2) has at least one positive solution.*

Proof Let

$$R(C) = \int_0^\infty B(a) \int_0^a e^{-(\mu+\gamma)(a-s)} G(C,s)ds$$

then $R(C)$ is continuous on C. If we let

$$\bar{C} = \inf\{\hat{C} \in (0,C_0) : R(C) > 1 \text{ for all } C \in (\hat{C}, C_0]\},$$

then $0 \leq \bar{C} < C_0$, since $R(C_0) > 1$.

Case 1. If $\bar{C} = 0$ then

$$f(C_0) = \frac{\mu C_0}{K - C_0} = r_0 = r(0) > r(\frac{U_{C_0}}{C_0}).$$

Lemma 3.2 yields

$$\lim_{C\to 0} r(\frac{U_C}{C}) \geq r(\rho) > r(1) \geq 0 = \lim_{C\to 0} \frac{\mu C}{K_C} = f(0).$$

Therefore, there is at least a $C_* \in (0, C_0)$ such that

$$r(\frac{U_{C_*}}{C_*}) = f(C_*).$$

Case 2. If $\bar{C} > 0$ then continuity implies that $R(\bar{C}) = 1$ and that

$$\lim_{C \to \bar{C}} u_C = 0.$$

Hence $f(C_0) > r(\frac{U_{C_0}}{C_0})$ and

$$f(\bar{C}) = \lim_{C \to \bar{C}} \frac{\mu C}{K - C} < \frac{\mu C_0}{K - C_0} = r(0) = \lim_{C \to \bar{C}+} r(\frac{U_C}{C}).$$

Consequently, there is a $C_* \in (\bar{C}, C_0]$ such that

$$\frac{\mu C_*}{K - C_*} = r(\frac{U_{C_*}}{C_*}).$$

Remark If $r(1) > 0$ then Lemma 3.2 is not needed in the proof of Theorem 3.3.

Corollary 3.4 *Suppose $R_0 > 1$ then System (1.4) has at least an endemic equilibrium.*

The proof can be found in Huang and Castillo-Chavez (2001).

Endemic equilibria when $R_0 < 1$

The ODE (non-age structured) model of Castillo-Chavez and Hadeler (1995) supports multiple endemic equilibria when $R_0 < 1$ but its epidemiological structure is more complex than in our age structured model. The assumption of age-independent rates and integration over all age classes leads to an ODE model that is not capable of supporting multiple endemic equilibria. It is therefore of some interest to determine whether or not our age structured model can indeed support endemic equilibria when $R_0 < 1$. To show that Model (1.4) can support positive equilibria when $R_0 < 1$ we need the following theorem which is stated without proof:

Theorem 4.1 *Given $B > 0$. For $\alpha > 0$ let $\hat{R}(\alpha)$ be defined by*

$$\hat{R}(\alpha) = \frac{\int_0^\infty B(a) \int_0^a e^{-\sigma(a-s)} B(s) e^{-\mu s}(1 - e^{-\alpha s}) ds da}{\int_0^\infty B(a) e^{-\mu a}(1 - e^{-\alpha a}) da}. \tag{4.1}$$

If there is an $\alpha_0 > 0$ such that $\frac{d\hat{(}\alpha_0)}{d\alpha} < 0$ then there exists a recruitment function r such that r is decreasing and the corresponding reproductive numver R_0 is less than 1 but yet, there exists an endemic equilibrium.

Example Let $B(a) = ae^{-\eta a}$ where $\eta > 0$. Hence, B is bounded, positive and $\lim_{a\to\infty} B(a) = 0$. By the definition (4.1) we have

$$\frac{d\hat{R}(a)}{d\alpha} = F(\alpha)[V(\alpha) - W(\alpha)],$$

where

$$\begin{aligned}
F(\alpha) &= \frac{1}{[\int_0^\infty B(s)e^{-\mu s}(1-e^{-\alpha s})ds]^2}, \\
V(\alpha) &= (\int_0^\infty [\int_s^\infty B(a)e^{-\sigma(a-s)}da]sB(s)e^{-(\mu+\alpha)s}ds) \int_0^\infty B(a)e^{-\mu a}(1-e^{-\alpha a})da, \\
W(\alpha) &= (\int_0^\infty [\int_s^\infty B(a)e^{-\sigma(a-s)}da]B(s)e^{-\mu s}(1-e^{-\alpha s})ds) \int_0^\infty B(a)ae^{-(\mu+\alpha)a}da.
\end{aligned}$$

Let $\sigma = \alpha = 2$, $\mu = 1$. A straightforward computation gives

$$\begin{aligned}
V(2) &= \frac{2(15+8\eta)(4+2\eta)}{(2+\eta)^2(2\eta+3)^3(3+\eta)^2(2\eta+3)(1+\eta)^2}, \\
W(2) &= \frac{2(5+4\eta)(2\eta+3)^3 - 2(7+4\eta)(2\eta+1)^3}{(2+\eta)^2(2\eta+3)^3(3+\eta)^2(2\eta+1)^3(3+\eta)}.
\end{aligned}$$

If we now let $\eta = 0$ then

$$V(2) - W(2) = \frac{2[15\cdot 4 - 128]}{2^3\cdot 3^3\cdot 3^3} < 0.$$

Therefore it follows from Theorem 4.1 that we can find a decreasing recruitment function such that its corresponding system has a stable disease free distribution, as well as an endemic equilibrium.

4. Conclusion

For natural selection to "advance" opportunities must be present. The growth of cities, the effects of mass transportation and international travel, the development of antibiotics, vaccines and other forms of treatment, the changes on values and costumes driven in part by globalization and modes of mass information are but some of the factors that make the study of disease dynamics challenging, complex and interesting. Every pressing question immediately brings to the forefront a multitude of choices: What should it be our epidemiological unit? What is the appropriate temporal or geographical scale? Does behavior matter? Is the population under study really isolated?

Clearly, one cannot assume the existence of a fixed landscape in the study of disease evolution.

In the study of disease emergence or re-emergence, it seems that one must be aware of the possibilities. Such possibilities are often encoded in the dynamical landscapes associated with models for the spread of a particular disease (landscapes are also closely connected to mechanisms). It seems to us that the occurrence of backward (subcritical) bifurcations, the existence of multiple infected stationary states, and hysteresis phenomena (including abrupt changes in disease prevalence levels) are but a few of the components that are supportive of disease reemergence. Models that generate this type of landscapes must be understood since they provide useful insights in the study of disease re-emrgence and evolution (see Castillo-Chavez et al. 1989, Huang et al. 1992, Hadeler et al. 1995 and Feng et al. 2000).

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References

Baldwin J.D. and J.I. Baldwin, (1988), *Factors affecting AIDS-related sexual risk-taking behavior among college students.* J. Sex Research **25**, 181-196.

Blythe, S.P., F. Brauer and C. Castillo-Chavez, (1997), *Demographic Recruitment in Sexually Transmitted Disease Model.* Proc. First World Congress on Computational Medicine, Public Health and Biotechnology Part II Austin, TX, 1994. Ed. Matthew Witten. Series in Mathematical Biology and Medicine **5**, 1438-1457.

Blythe, S.P., F. Brauer, . Castillo-Chavez and J. X. Velasco-Hernandez, (1993a), *Models for sexually transmitted diseases with recruitment.* Biometrics Unit Technical Report BU-1193-M, Cornell University.

Blythe, S.P., K. Cooke and C. Castillo-Chavez, (1993b), *Autonomous risk-behavior change, and non-linear incidence rate, in models of sexually transmitted diseases.* Biometrics Unit Technical Report BU-1048-M, Cornell University, 1993b.

Brauer, F., C. Castillo-Chavez and J.X. Velasco-Hernandez, (1998), *Recruitment into Core Group and its effect on the spread of a sexually transmitted disease.* Advances

in Mathematical Population Dynamics - Molecules, Cells, and Man (O. Arino, D. Axelrod, M. Kimmel, (eds), World Scientific Press, 477 - 486, 1998.

Busenberg, S. and C. Castillo-Chavez, (1991), *A general solution of the problem of mixing subpopulations, and its application to risk- and age-structure epidemic models for the spread of AIDS,* IMA J. of Math. Appl. Med. Biol., 8, pp. 1-29.

Busenberg, S. and K. P. Hadeler, (1990), *Demography and epidemics.* Math. Biosc. **101**, 63-74.

Castillo-Chavez, C. ed. (1989), *Mathematical and Statistical Approaches to AIDS Epidemiology*, vol.83 of Lecture Notes in Biomathematics, Springer-Verlag, Berlin.

Castillo-Chavez, C., K. Cook, W. Huang, and S.A. Levin, (1989), *Results on the dynamics for models for the sexual transmission of human immunodeficiendy virus.* Appl. Math. Lett. **2(4)**, 327-331.

Castillo-Chavez, C, Z. Feng and W. Huang, (2001), *On the computation of R_0 and its role on global stability*, This volume.

Castillo-Chavez, C., J.X. Velasco-Hernandez, and S. Fridman, (1994), *Modeling contact structures in Biology.* In: *Frontiers of Theoretical Biology,* S.A. Levin (ed.), Lecture notes in Biomathematics, 100. Spring-Verlag, New York.

Curran, J. W., H.W. Jaffe, A.M. Hardy, M.W. Morgan, R.M. Selik, T.J. Dondero, (1988), *Epidemiology of infection and AIDS in the United States.* Science **293**, 610-616.

Diekmann, O., J.A.P. Heesterbeek, and J.A.J. Metz, (1990), *On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous population,* J. Math. Biol. **28**, 365-382

Diekmann, O. and J. A. P. Heesterbeek, (2000), *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation.* Wiley, New York.

Evans, B. A., K.A. McLean, S.G. Dawson, et al., (1989), Trends in sexual behavior and risk factors for the HIV infection among homosexual men, 1984-1987. Brit. Med. J. **298**, 215-218.

Feng, Z., C. Castillo-Chavez, and A. Capurro (2000), *A model for TB with exogenous reinfection*, Theo. Pop. Biol. **578**, 237-247.

Fineberg, H. V. (1988), *Education to prevent AIDS: prospects and obstacles.* Science **239**, 592-596.

Hadeler, K.P., R. Waldstätter and A. Wörz-Busekros, (1988), *Models for pair formation in bisexual populations.* J. Math. Biol. **26**, 635-649.

Hadeler, K.P., (1992), Periodic solutions of homogeneous equations. J.Diff.Equ. 95, 183-202.

Hadeler, K.P. and C. Castillo-Chavez, (1995), *A core group model for disease transmission* , Math. Biosci. **128**, 41-55.

Hethcote H. W. and J.W. Van Ark, (1987), *Epidemiological models with heterogeneous populations: Proportionate mixing, parameter estimation and immunization programs*, Math. Biosci., 84, pp.85–118.

Hethcote H. W. and J.W. Van Ark, (1992), *Modeling HIV Transmission and AIDS in the United States*, vol.95 of Lecture Notes in Biomathematics, Springer-Verlag, Berlin.

Hethcote, H. W. and J.A. Yorke, (1984), *Gonorrhea: Transmission Dynamics and Control.* Lecture Notes in Biomathematics **56**, Springer-Verlag.

Hsieh, Y. and K. Cooke, (2000), *Behaviour change and treatment of core groups: its effect on the spread of HIV/AIDS.* IMA J. of Math. Applied to Medicine and Biology **17**, 213–241.

Huang, W., K.L. Cooke, S. and C. Castillo-Chavez, (1992), *Stability and Bifurcation for a multiple-group model for the dynamics of HIV-AIDS transmission*, SIAM J. Appl. Math. **52**, 835-854.

Huang, W. and C. Castillo-Chavez, (2001),*On the role of disease driven recruitment on the dynamics of STDs in age-structured populations* , in preparation.

Martin, J. L., (1987), *The impact of AIDS in gay male sexual behavior patterns in New York City.* Am. J. of Pub. Health **77** 578-581.

McKusick, L., J.A. Wiley, T.J. Coates, R. Stall, B. Saika, S. Morin, C.K. Hortsman and M.A. Conant, (1985), *Reported changes in the sexual behavior of men at risk for AIDS, San Francisco, 1983-1984: the AIDS behavioral research project.* Public Health Reports **100**, 622-629.

Saltzman, S.P., A.M. Stoddard, J. McCusker, M.W. Moon and K.H. Mayer, (1987), *Reliability of self-reported sexual ehavior risk factors for HIV in homosexual men.* Public Health Report **102**, 692- 697.

McClam, E., (2001), *"Syphilis Outbreak alarms officials"*, Ithaca Journal, Section A.,

page 10A, February 23, 2001.

Scalia-Tomba, G., (1991), *The effect of structural behavior change on the spread of HIV.* Math. Bios. **107**, 547–556.

Shechter, M.T., K.J.P. Craib, B. Willoughby, B. Douglas, W. Alastair McLeod, M. Maynard, P. Constance and M. O'Shaughnessy, (1988), *Patterns of sexual behavior and condom use in a cohort of homosexual men.* Amer. J. Publ. Health **78**, 1535-1538.

Shilts, R., (1987), And the band played on. San Martin's press, New York

van Griensven, G.J.P., de Vroome, E.M.M, Goudsmit, J., Coutinho, R.A. (1989a), *Changes in sexual behavior and the fall in incidence of HIV infection among homosexual men.* Br. Med.J. **298**, 218-221.

van Griensven, G.J.P, E.M.M. de Vroome, R.A.P. Tielman, J. A. Goudsmit, A. P. de Wolf F., J. van der Noordaa and R. A. Coutinho (1989b), *Effect of human immuno-deficiency virus (HIV) antibody knowledge on high-risk sexual behavior with steady and non-steady partners among homosexual men.* Am.J. Epidem. **129**, 596-603.

Velasco-Hernandez, J. X., F. Brauer and C. Castillo-Chavez (1996), *Effects of Treatment and Prevalence-dependent Recruitment on the Dynamics of a fatal disease.* IMA Journal of Math. Medicine and Biology, **13** 3, 175-192.

Velasco-Hernandez J.X. and Y. Hsieh (1994), *Modelling the effect of treatment and behavioral change in HIV transmission dynamics.* J. Math Biol. **32**, 233–249.

Webb, G. F., (1985), *Theory of Nonlinear Age-dependent Population Dynamics*, Marcel Dekker, New York.

Wiktor, S.Z., R.J. Biggar, M. Melby, P. Ebbesen, G. Colclough, R. Di Gioia, W.C. Sanchez, R.J. Grossman, J. Goedert, (1990), *Effects of knowledge of human immunodeficiency virus infection status on sexual activity among homosexual men.* J.of AIDS **3**, 62-68.

Wilkenstein, W. Jr., J.A. Wiley, N.S. Padian, M. Samuel, S. Shiboski, M. S. Ascher and J. A. Levy, (1988), *The San Francisco Men's Health Study, continue decline in HIV seroconversion rates among homosexual/bisexual men.* Am. J. Public Health **78**, 1472-1474.